Case report

Acute Intestinal Obstruction Due to Intestinal Anisakiasis Resolved with Conservative Therapy

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Abstract

Intestinal anisakiasis is rarely diagnosed because it is thought to be uncommon and is poorly recognized. It produces severe abdominal pain and an inflammatory reaction often resulting in reactive intestinal obstruction, which is sometimes treated with an unnecessary laparotomy as acute abdomen or intestinal obstruction. We reported a 58-year-old female with acute intestinal obstruction caused by intestinal anisakiasis, which resulted in a self-limiting clinical course. The diagnosis was based on a history of recent ingestion of raw fish and abdominal computed tomographic findings of partial thickening of the intestinal wall accompanied by focal luminal narrowing with ascites. In spite of the severity of the abdominal pain, the bowel obstruction induced by inflammation and edema was resolved with conservative treatment after three weeks. Accordingly, intestinal anisakiasis was considered in the differential diagnosis of intestinal obstruction, which can be treated with conservative therapy.

Key words: Intestinal anisakiasis, intestinal obstruction, computed tomography, conservative therapy, abdominal pain

Introduction

Anisakiasis (Anisakidosis) is a parasitic disease caused by accidental ingestion of Anisakis larvae. Anisakis pathology is due mainly to two mechanisms, allergic reactions (from isolated urticaria and angioedema to life-threatening anaphylaxis shock associated with gastrointestinal symptoms or 'gastroallergic anisakiasis') and direct tissue damage due to invasion of the gut wall, development of eosinophilic granuloma or perforation. Judging from the involved sites, the clinical forms of direct tissue damage have been classified into three types, gastric, intestinal and ectopic anisakiasis1–3). According to a series of 15,715 cases of anisakiasis reported by Ishikura, gastric anisakiasis accounts for 95.6% of cases, intestinal anisakiasis accounts for 4.1% of cases and other sites accounts for 0.3% of case4). Intestinal anisakiasis produces diffuse severe abdominal pain and an inflammatory reaction often resulting in reactive intestinal obstruction. The pathogenesis is caused not only by the physical action of perforation by the larvae, but also by the associated allergic Arthus-type and/or anaphylaxis reactions, which induce severe abdominal pain and inflammation5–8). The diagnosis of intestinal anisakiasis, however, has rarely been made because it is thought to be uncommon and is poorly recognized in regard to its clinical and laboratory findings. It is also difficult to identify Anisakis larvae without bowel surgery9,10). We herein report a case of intestinal obstruction due to anisakiasis, which improved with conservative therapy in a three-week clinical course.

Case Report

A 58-year-old female presented to our hospital complaining of abdominal pain. She had been well until the day before admission, when she had mild abdominal pain that became persistent with nausea. She had eaten pickled cuttlefish five days before the onset. She denied vomiting and diarrhea. Her medical history was remarkable for hypertension and a previous appendectomy at age 21. She does not smoke or drink. On physical examination, the abdomen revealed generalized distention, a scar at the right lower quadrant, hyperactive bowel sounds and mild tenderness in the whole abdomen without rebound tenderness (Blumberg’s sign) and rigidity. The remainder of the physical
examination was unremarkable. Laboratory examination showed mild leukocytosis of 10000/µL with 85.2% neutrophils and 4% eosinophils, elevated C-reactive protein of 6.41 mg/dL, BUN of 22 mg/dL and amylase of 35 IU/L. Other blood screening tests, urinalysis and an electrocardiogram were normal. A chest X-ray was normal including no free air beneath the diaphragm, but a plain standing X-ray of the abdomen showed gaseous distension of the small bowel (Figure 1).

The patient was admitted with the diagnosis of acute intestinal obstruction (small bowel obstruction). Contrast enhanced computerized tomographic (CT) scanning of the abdomen demonstrated marked swelling of a partial segment of the symmetric wall at the level of the jejunum with diffuse contrast enhancement and luminal narrowing (Figure 2a), as well as small bowel distension with intraluminal fluid collection at the level of the oral side of the lesion (Figure 2b). Collapse of the ileum and large bowel with ascites accumulation at the Douglas’ pouch was also noted. Conservative management with a long nasogastric tube was chosen, as the patient’s status did not indicate strangulation of the bowel. It was possible to insert the tube 160 cm past the incisor teeth, which positioned it 50 cm from the ligament of Treitz. Consequently, approximately 850 mL of intestinal fluid was drained over the course of half a day. Her pain gradually ameliorated. On day 6 after admission, an abdominal CT revealed improvement of the intestinal swelling. A small bowel series showed a narrowed jejunum segment without any obstruction (Figure 3). The result of a test for Anisakis-specific IgA on her fourth day in hospital revealed an elevated value of 1.80 IU (normal range < 1.50). On day 8, the patient started to eat solid food, and she was ultimately discharged without complication on day 21.

Figure 1  A plain standing X-ray of the abdomen on admission demonstrated gaseous distension of the small bowel.

Figure 2a  Contrast enhanced CT of the abdomen on admission disclosed marked thickening of a partial segment of the symmetric wall at the level of the jejunum with diffuse contrast enhancement and luminal narrowing.

Figure 2b  Small bowel distension with intraluminal fluid collection at the level of the oral side of the lesion was observed.
Discussion

Anisakiasis can be confirmed by detecting morphologic characteristics of the whole worm at endoscopy. Different from gastric anisakiasis, intestinal anisakiasis is difficult to diagnose because the small intestine is an inaccessible zone for endoscopy and the abdominal symptoms are nonspecific. As a result, some patients with intestinal anisakiasis have undergone surgical exploration after diagnosis of an acute abdomen or intestinal obstruction. Ishikura et al. reported that intestinal anisakiasis was correctly diagnosed postoperatively in 23% of cases as acute appendicitis in 38% of cases, as intestinal obstruction in 12% of cases, and as acute celiotomy in 10% of cases. Some reports argue that an early surgical approach should be avoided in cases where an intestinal obstruction suggests the possibility of intestinal anisakiasis. Since anisakis larvae only survive for a few days in the intestinal tracts of humans, acute inflammation subsides within 2 to 3 weeks, and therefore empirical treatment using decompression with nasogastric tubing is recommended, as observed in our case. However, there are a reported case of strangulating intestinal obstruction from eosinophilic granulomas and two cases of severe long segmental stenosis, all of which were caused by intestinal anisakiasis and required surgical therapy.

Intestinal anisakiasis is diagnosed by clinical history and indirect findings from imaging studies. Gastric anisakiasis usually develops several hours after ingestion of raw fish, whereas the onset of intestinal anisakiasis is delayed from 1 to 7 days. Anamnesis from raw or undercooked fish ingestion before the onset of digestive symptoms has been reported with a sensitivity of 98% and a specificity of 74% for intestinal anisakiasis. Most laboratory findings suggest only nonspecific acute inflammatory changes. Eosinophilia is usually not present in patients with gastric or intestinal anisakiasis, as seen in our case. Immunological examination (e.g., Anisakis-specific IgE, IgA, IgG) is specific but takes time and fails to be helpful for acute conditions.

Typical CT findings include thickening of a partial segment of the symmetric wall with luminal narrowing, diffuse contrast enhancement in the involved segment and ascites, as observed in our case. According to a series of 18 cases of intestinal anisakiasis reported by Shirahama, the stenotic segment was 10–40 cm long, with a mean value of 19 cm, and the terminal ileum was involved in 70% of the cases. Ultrasound findings also suggest accumulation of ascites, dilatation of the small intestine with fluid accumulation and marked localized edema of Kerckring’s folds or wall thickening. Based on the CT findings, the differential diagnoses of the disease included adenocarcinoma of the small bowel and inflammatory bowel diseases. Intestinal adenocarcinoma usually involves only a short segment and presents on CT as an eccentric focal mass or as a circumferential asymmetric and irregular thickening of the bowel wall.

In summary, we diagnosed intestinal anisakiasis causing acute intestinal obstruction, on the basis of the patient’s history and CT findings, which was successfully managed with conservative measures.

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References